# SPECKLE TRACKING ECHOCARDIOGRAPHY MECHANICAL DISPERSION AS A RISK MARKER FOR VENTRICULAR ARRHYTHMIA IN HYPERTROPHIC CARDIOMYOPATHY AND ITS CORRELATION WITH FIBROSIS ON CARDIAC MRI, SUDDEN CARDIAC DEATH SCORES

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# INTRODUCTION

Hypertrophic cardiomyopathy (HCM) is the most common monogenetic cardiovascular disorder, with a prevalence of 1 per 500 persons in the general population (Maron et al., 1995). Clinical diagnosis of HCM is based on hypertrophied, nondilated left ventricle – which is identified using Echocardiography or Cardiac Magnetic Resonance (CMR) in the absence of another cardiac, systemic, metabolic, or syndromic disease (Authors/Task Force members et al., 2014), (Gersh et al., 2011), (Maron, 2018). HCM is the most common cause of sudden cardiac death (SCD) in the young, is responsible for progressive heart failure and disability in a wide range of ages, and is associated with a significant risk for atrial fibrillation and embolic stroke. Mortality rates in Adults have reduced from 1.5 % per year to 0.5 % per year with the use of contemporary treatment and intervention like Implantable cardioverter defibrillator (ICD) (Maron et al., 1999), (Rowin et al., 2013). Various Sudden Cardiac death scores were designed to assess the risk of SCD so patients can benefit from ICD Implantation. ICD is indicated as primary prophylaxis if the European Society of Cardiology (ESC) SCD risk score $\geq$ 6, and as secondary prophylaxis in patients with prior resuscitated cardiac arrest due to Ventricular Tachycardia (VT)/ Ventricular Fibrillation(VF) or prior VT/VF causing Syncope or hemodynamic compromise (Authors/Task Force members et al., 2014). Guidelines leave some patients at risk of SCD, and some receive ICDs without benefitting them (Leong et al., 2018). Hence the necessity to search for other reliable markers of SCD. Strain Echocardiography parameters like Global longitudinal strain (GLS), Mechanical dispersion (MD) were

found to be associated with the prediction of Ventricular Arrhythmia and the extent of fibrosis (Haland et al., 2016).

## SCOPE OF THE STUDY

Identifying HCM patients prone to Ventricular Arrhythmia is vital to prevent the risk of SCD. There is a need to identify appropriate risk markers for Ventricular Arrhythmia. MD appears promising in identifying patients with HCM prone to VA. Because of the lack of Studies, this study is undertaken to evaluate the association between strain echocardiographic parameter MD and SCD Risk scores, Ventricular arrhythmias, and extent of fibrosis.

# MATERIALS AND METHODS

## *3.1 AIM OF THE STUDY*

- Study the relationship between mechanical dispersion and ventricular Arrhythmia in hypertrophic cardiomyopathy

## *3.2 OBJECTIVES*

1) Study the relationship between mechanical dispersion and ventricular Arrhythmia in hypertrophic cardiomyopathy
2) Study the relationship between Global longitudinal strain and ventricular Arrhythmia in hypertrophic cardiomyopathy
3) Study risk markers (ACC/AHA risk markers) and risk scores ( ESC 5-year SCD risk score) for SCD in HCM

## *3.3 METHODOLOGY*

**Study Design:** Prospective, Cross-sectional study

**Study Setting**: Department of Cardiology, Sree Chitra Tirunal Institute for Medical Sciences and Technology (SCTIMST), Trivandrum

**Study Subjects:** Consecutive HCM patients from Jan 2020 were selected from the HCM registry, Department of cardiology. 120 HCM patients are screened, and after exclusion criteria, 56 HCM patients are included in the study

**Inclusion criteria:**

Patients with Clinical diagnosis of HCM based on HCM guidelines

HCM is defined by a wall thickness ≥15 mm in one or more LV myocardial segments—as measured by Echocardiography that is not explained solely by loading conditions (Authors/Task Force members et al., 2014).

**Exclusion criteria:**

HCM Patient

1) With Coronary Artery Disease
2) With LV dysfunction (LVEF < 55%)
3) Myocardial hypertrophy of other causes such as
    a. Severe valvular disease
    b. Myocardial storage disease
    c. Uncontrolled Hypertension
4) Patients not in Sinus rhythm during Speckle tracking Echocardiography
    a. Atrial Fibrillation
    b. Predominantly Paced rhythm
5) Patients not willing to give consent for the study

56 HCM patients are categorized into two groups, 42 patients in HCM without ventricular Arrhythmia (VA) and 14 patients in HCM with Ventricular Arrhythmia group.

**Ventricular Arrhythmia (VA)**

The presence of VA is defined as

i) previous aborted cardiac arrest
ii) documented sustained ventricular tachycardia (VT)
iii) NSVT during Holter or ICD monitoring

NSVT is defined as runs of ≥3 ventricular beats and < 30 s duration with a heart rate >120 bpm.

Clinical examination, Echocardiography, 24 Holter monitoring, and CMR findings are collected from the patient's Electronic Medical Records.

Holter examination was done in HCM patients, and on Holter, if they were not found to have NSVT/VT/VF, they were included in HCM without VA group.

Speckle tracking Echocardiography for 2D Strain and Mechanical dispersion was done on all selected patients.

**SCD Risk Scores:**

***ESC SCD Risk factors*** (Authors/Task Force members et al., 2014)**:**

1) Age
2) Non-sustained ventricular tachycardia (NSVT): NSVT is defined as ≥3 consecutive ventricular beats at ≥120 BPM lasting <30 seconds during ambulatory ECG monitoring
3) Maximum left ventricular wall thickness: maximum wall thickness of ≥30 mm on TTE
4) Family history of sudden cardiac death at a young age: 1 or more 1°relative have died suddenly aged < 40 years with or without a diagnosis of HCM or SCD has occurred in 1°relative at any age with an established diagnosis of HCM
5) Syncope: Non-neurocardiogenic Syncope, for which there is no explanation after an investigation, is associated with an increased risk of SCD.
6) Left atrial diameter
7) Left Ventricular Outflow tract obstruction (LVOTO)

Probability of SCD at 5 years = $1 - 0.998^{\exp(\text{Prognostic index})}$

where Prognostic index = [0.15939858 x maximal wall thickness(mm)] - [0.00294271 x maximal wall thickness$^2$ (mm$^2$)] + [0.0259082 x left atrial

diameter (mm)] + [0.00446131 x maximal (rest/Valsalva) left ventricular outflow tract gradient (mm Hg)] + [0.4583082 x family history SCD] + [0.82639195 x NSVT] + [0.71650361 x unexplained syncope] - [0.01799934 x age at clinical evaluation (years)].

**Major SCD Risk Markers based on ACC-AHA** (Gersh et al., 2011) **and more recent data** (Rh et al., 2014)**,** (Rowin et al., 2017)**,** (Harris et al., 2006)**:**

1) Family History of HCM-related SCD: history of HCM-related sudden death in ≥1 first-degree or other relatives <50 years of age
2) Unexplained Syncope: Syncope (exclude neural mediated Syncope)
3) Nonsustained Ventricular Tachycardia (NSVT): NSVT is defined as ≥ three consecutive ventricular beats at ≥120 BPM lasting <30 seconds
4) Maximum LV Wall Thickness: Left Ventricular Hypertrophy (LVH) ≥ 30 mm
5) Extensive LGE: > 15% LV mass (Rh et al., 2014)
6) LV Apical Aneurysm (Rowin et al., 2017)
7) End Stage (Ejection fraction < 50%) (Harris et al., 2006)

**Echocardiography:**

i) Maximum wall thickness (MWT) was measured from all LV segments from base to apex of the left ventricle in Parasternal short axis view at Mitral, Mid LV, and Apical levels (Authors/Task Force members et al., 2014).

ii) Asymmetric septal hypertrophy is defined as septal to posterior free wall ratio > 1.3 (Afonso et al., 2008).

iii) LV end-diastolic diameter (LVEDD) and LV end-systolic diameter (LVESD) were measured in the Parasternal long axis view (Lang et al., 2015).

iv) Ejection fraction (EF) was calculated by modified Simpson's biplane method

v) Diastolic function was evaluated by trans mitral pulsed Doppler and average e′ from septal and lateral tissue Doppler samplings (Nagueh et al., 2016).

vi) Atrial diameter was determined by M-mode or 2D Echocardiography in the parasternal long-axis plane (Authors/Task Force members et al., 2014), and the atrial area was calculated as the average end-systolic area from apical four- and two-chamber views (Lang et al., 2015).

vii) left atrial volume (LAV) was calculated using the area length method, LAV corrected for body surface area gives left atrial volume index (LAVI) (Lang et al., 2015).

viii) Left ventricular outflow tract (LVOT) gradient was assessed only at rest, and a pressure gradient of ≥30 mmHg was defined as significant obstruction (Authors/Task Force members et al., 2014)

**2D Strain and Mechanical dispersion:**

Longitudinal strain by speckle tracking echocardiography was obtained from three apical views (Apical 4 Chamber view, Apical 2 Chamber View, Apical 3 Chamber view) at a frame rate of > 50/s.

Global longitudinal strain (GLS) is defined as an average of peak longitudinal strain from an 18 left Ventricle (LV) Segments model.

Time to peak strain was defined as the time from onset of Q/R wave on ECG to peak negative longitudinal strain during the entire cardiac cycle.

Mechanical dispersion is defined as the standard deviation of time to peak negative strain in 18 LV segments (Haugaa et al., 2010).

Echocardiography, Strain echocardiography was done in the Philips EPIQ 7c model by a single operator.

**Cardiac magnetic resonance (CMR) imaging**

CMR is performed with cine imaging with late gadolinium enhancement (LGE-CMR) in patients using 1.5 T units ( Magnetom Avanto fit, Siemens, Erlangen, Germany) using a phased array body coil. LGE is obtained with multiple short-axis slices covering the entire LV with a slice-to-slice increment of 10 mm, 10–20 min after intravenous injection of 0.2 mmol/kg gadopentetate dimeglumine. A breath-hold segmented magnetization-prepared turbo gradient echo sequence is used with an inversion time chosen to null normal myocardial signal.

Cardiac volumes and EF are calculated according to the biplane area length method from the two long-axis cine projections. For the calculation of LV mass, the endocardial and epicardial borders were manually drawn on short-axis cine images in end-diastole. The presence of LGE as a categorical value is first assessed by the radiologist blinded to the patient's clinical data. LGE quantification is performed using Circle Cvi 42 version 5.0. LGE is defined as areas with an adjusted grey-scale threshold ≥5 SD above the mean of normal myocardium, and these areas are

automatically traced in all slices by the software. Areas of identified LGE were summarized and quantified as a proportion of total LV myocardium (%LGE).

**Statistical analysis:**

Parametric data are presented as mean + standard deviation and compared by unpaired Student's t-test or by $\times^2$ or Fischer's exact test. % LGE is not normally distributed and presented as median (min-max) compared with non-parametric tests. The correlation between echocardiographic parameters and % LGE was assessed by linear regression analysis. Univariate logistic regression was used to identify markers for VA. Multivariate analyses were performed, including age and significant ($P < 0.05$) variables from the univariate analyses. Receiver operator characteristic (ROC) curves were created and the value closest to the upper left corner determined optimal sensitivity and specificity to discriminate between HCM patients with VAs. Likelihood ratios were used to test whether strain echocardiography improved the risk stratification of VA when added to the conventional SCD Risk stratification score (SPSS version 28, SPSS Inc., Chicago, IL, USA). Inter- and intra- observer variabilities were expressed by Intra class correlation coefficients. Two-sided values of $P \leq 0.05$ was considered statistically significant.

***ETHICAL JUSTIFICATION***

The present study entails an evaluation of Mechanical dispersion obtained by Strain Echocardiography to risk stratify patients of HCM prone to Ventricular Arrhythmia. As Echocardiography is a simple, non-invasive test compared to costly MRI, MD can be easily done on follow-up of patients also if found to be helpful to risk stratifying

patients of HCM. This study was undertaken because there is a paucity of studies in this area.

This study was approved by the Institutional Ethics Committee, SCTIMST Trivandrum.

# RESULTS

120 HCM patients were screened, and after excluding patients with Atrial fibrillation, LV dysfunction, and patients who did not turn out for ECHO evaluation, 56 patients of HCM were included in the study.

42 (75%) patients were included in HCM without VA group, 11 (20%) patients had NSVT on Holter, 3 (5%) patients had VT, and one patient in the VT group had Resuscitated cardiac arrest. Patients with NSVT and VT were included in HCM with VA group 14 (25%).

46 (82%) had ASH, 7 (12.5%) had Apical hypertrophy, 2 (3.6%) had concentric hypertrophy, and one (1.8%) had midventricular hypertrophy**.**

**Baseline characteristics of the HCM cohort**

***Table 1: Baseline characteristics of the HCM cohort included in the study and both the groups***

| | HCM (n=56) | HCM without VA(n=42) | HCM with VA (n = 14) | P |
|---|---|---|---|---|
| Mean age (yrs.) | 50 ± 14 | 48 ± 15 | 55 ± 9 | 0.115 |
| Mean age at presentation (yrs.) | 46 ± 15 | 46 ± 15 | 45 ± 13 | 0.904 |
| Female | 18 (32%) | 17 (40%) | 1 (7%) | 0.023 |
| Male | 38 (68%) | 25 (60%) | 13 (93%) | 0.023 |
| Weight (kg) | 68 ± 11 | 67 ± 9 | 71 ± 14 | 0.212 |
| Height (cm) | 158 ± 23 | 156 ± 26 | 164 ± 7 | 0.262 |
| BSA (m2) | 1.7 ± 0.16 | 1.7 ± 0.1 | 1.7 ± 0.1 | 0.417 |

| | | | | |
|---|---|---|---|---|
| BMI (kg/m2) | 26 ± 3.4 | 25.6 ± 3.3 | 26 ± 3.8 | 0.648 |
| SBP (mm Hg) | 130 ± 20 | 132 ± 21 | 121 ± 14 | 0.066 |
| DBP (mm Hg) | 76 ± 10 | 77 ± 10 | 72 ± 8 | 0.076 |

p-value is between HCM with & without VA groups

38(68%) of patients were male in the HCM cohort, and almost 13 (93%) were male in the HCM with VA group.

**ECG parameters**

***Table 2: ECG parameters in HCM patients & both the groups***

| | HCM (n=56) | HCM without VA(n=42) | HCM with VA (n = 14) | p |
|---|---|---|---|---|
| HR/mt | 71 ± 17 | 70 ± 10 | 74 ± 28 | 0.632 |
| PR (ms) | 183 ± 98 | 184 ± 111 | 180 ±37 | 0.904 |
| QRSD (ms) | 92 ± 17 | 88 ±12 | 102 ± 25 | 0.072 |
| QT (ms) | 422 ± 36 | 418 ± 30 | 435 ±51 | 0.133 |
| QTc (ms) | 443 ± 26 | 442 ± 20 | 447 ± 37 | 0.546 |
| LVH | 48 (85%) | 39 (92%) | 9 (64%) | 0.018 |

p-value is between HCM with & without VA groups

QRS duration was more in HCM with the VA group, and LVH was more in HCM without VA.

**Holter findings**

***Table 3: Holter findings in HCM patients & both the groups***

| | HCM (n=56) | HCM without VA (n=42) | HCM with VA (n=14) | |
|---|---|---|---|---|
| Average heart rate/mt | 66 ± 8 | 67 ± 6 | 65 ± 11 | 0.598 |
| Minimum heart rate/mt | 47 ± 8 | 47 ± 7 | 48 ± 9 | 0.715 |

| | | | | |
|---|---|---|---|---|
| Maximum heart rate/mt | 115 ±22 | 116 ± 21 | 111 ± 24 | 0.583 |
| APC % in holter | 0.023 ± 0.1 | 0.029 ± 0.1 | 0 | - |
| VPC % in holter | 1.3 ± 4.7 | 0.7 ± 4 | 3.7 ± 7 | 0.274 |

p-value is between HCM with & without VA groups

11 (20%) patients had NSVT on Holter of the whole HCM cohort.

**Symptoms, family history**

***Table 4: Symptoms & family history in HCM patients & both the groups***

| | HCM (n=56) | HCM without VA(n=42) | HCM with VA (n = 14) | P |
|---|---|---|---|---|
| Dyspnoea | 22 ( 40%) | 19 (45%) | 3 (21%) | 0.1 |
| Syncope | 19 (34%) | 11 (26%) | 8 (57%) | 0.034 |
| Chest pain | 17 (30%) | 15 (35%) | 2 (14%) | 0.131 |
| Palpitation | 17 (30%) | 12 (28%) | 5 (35%) | 0.615 |
| Fatigue | 3 (5%) | 2 (4.8%) | 1 (7.1%) | 0.732 |
| Res SCD | 1 (1.8%) | 0 | 1 (7.1%) | 0.08 |
| F/h/o HCM | 11 (20%) | 6 (14%) | 5 (35%) | 0.08 |
| F/h/o HCM SCD 1 degree relative at any age | 6 (11%) | 4 (9.5%) | 2 (14%) | 0.618 |
| F/h non HCM SCD < 40 years age | 0 | 0 | 0 | |
| HTN | 9 (16%) | 9(21%) | 0 | **0.05** |
| DM | 1 (1.8%) | 1 (2.4%) | 0 | 0.56 |
| NYHA I | 23 (41%) | 13 (31%) | 10 (71%) | 0.026 |
| NYHA II | 31 (55%) | 27 (64%) | 4 (28%) | 0.026 |
| NYHA III | 2 (3.6%) | 2 (4.8%) | 0 | 0.026 |
| NYHA IV | 0 | 0 | 0 | - |

p-value is between HCM with & without VA groups

Dyspnea is the most common symptom in the cohort, followed by Syncope. Syncope and F/h/o HCM were more common in HCM with VA. Most of the patients in the cohort are in NYHA class II (31 patients 55%), followed by NYHA class I (23

patients 41%). Most patients in HCM with VA are in NYHA Class I compared to most patients in HCM without VA in NYHA Class I.

**Drug history**

***Table 5: Drugs in HCM patients & both the groups***

| | HCM (n=56) | HCM without VA(n=42) | HCM with VA (n = 14) | P |
|---|---|---|---|---|
| Beta-blockers | 46 (82%) | 35 (83%) | 11 (78%) | 0.687 |
| CCBs | 6 (11%) | 5 (12%) | 1 (7%) | 0.618 |
| Cordarone | 6 (11%) | 0 | 6 (43%) | **< 0.01** |
| Ranolazine | 4 (7%) | 3 (7%) | 1 (7%) | 1 |

p-value is between HCM with & without VA groups

46 (82%) patients with HCM were on Beta-blockers.

**ICD implantation findings**

***Table 6: ICD data in HCM patients & both the groups***

| | HCM n=56 | HCM without VA(n=42) | HCM with VA (n = 14) | P |
|---|---|---|---|---|
| Placed | 17 (30%) | 4 (9.5%) | 13 (93%) | **< 0.01** |
| Primary prophylaxis | 14 (25%) | 4 (9.5%) | 10 (71%) | 0.541 |
| Secondary prophylaxis | 3 (5%) | 0 | 3 (21%) | 0.541 |
| ICD Shock | 2 (3.6%) | 0 | 2 (14%) | 0.574 |

p-value is between HCM with & without VA groups

17 (30%) patients in the HCM cohort underwent ICD implantation, and four (9.5%) patients in HCM without VA underwent ICD implantation against

13 (93%) patients in HCM with VA group. In HCM with VA group, 10 (71%) patients had NSVT on holter and underwent ICD implantation as Primary Prevention, three (21%) underwent ICD implantation as Secondary prevention, out of these three, two had clinical VT, and one patient had a history of resuscitated cardiac arrest**.** Two patients with ICD implantation for secondary prevention had a shock for documented VT, and no shock therapy was delivered in patients with ICD implantation for primary prevention.

**Echocardiography parameters**

***Table 7: Echocardiography parameters in HCM patients & both the groups***

| | HCM (n=56) | HCM without VA(n=42) | HCM with VA (n = 14) | P |
|---|---|---|---|---|
| LVEDD (mm) | 39 ± 6.5 | 38 ± 6 | 41 ± 7 | 0.243 |
| LVESD (mm) | 23 ± 5.4 | 23 ± 5 | 24 ± 7 | 0.656 |
| IVS (S) | 24 ± 6.7 | 24 ± 7 | 24 ± 5 | 0.865 |
| IVS (D) | 20 ± 6.2 | 20 ± 6 | 21 ± 6 | 0.769 |
| PW (S) | 16 ± 4.4 | 16 ± 4 | 16 ± 5 | 0.551 |
| PW (D) | 12.3 ± 3.5 | 13 ± 3 | 12 ± 4 | 0.462 |
| MWT (mm) | 22 ± 6.5 | 22 ± 7 | 23 ± 5 | 0.591 |
| LVEF m mode (%) | 70 ± 8 | 71 ± 8 | 60 ± 7 | 0.347 |
| LVEDV (ml) | 64 ± 24 | 62 ± 22 | 74 ± 28 | 0.120 |
| LVESV (ml) | 24 ± 12 | 24 ± 12 | 29 ± 12 | 0.226 |
| LVEF Simpson (%) | 61 ± 7.2 | 61 ± 8 | 61 ± 5 | |
| LA (mm) | 39 ± 6 | 39 ± 6 | 40 ± 6 | 0.541 |
| LAESV | 56.5 ± 22 | 56 ± 23 | 57 ± 18 | 0.794 |
| LAESVI | 33.7 ± 13.8 | 34 ± 14 | 33 ± 10 | 0.808 |
| AO (mm) | 28 ± 4 | 29 ± 4 | 29 ± 4 | 0.986 |

| | | | | |
|---|---|---|---|---|
| LV Mass (gm) | 265 ± 96 | 261 ± 98 | 278 ± 93 | 0.567 |
| LV Mass I ($gm/m^2$) | 154± 53 | 153 ± 53 | 158 ± 56 | 0.746 |
| LVOT grad | 33 ± 38 | 40 ± 40 | 14 ± 23 | 0.004 |
| LVOT grad Val | 53 ± 27 | 64 ± 20 | 25 ±-28 | 0.029 |
| TR grad | 21 ± 9 | 23 ± 9.5 | 16 ± 7.4 | 0.034 |
| E | 84 ± 28 | 87 ± 29 | 76 ± 23 | 0.202 |
| A | 69 ± 31 | 74 ± 33 | 55 ± 18 | 0.051 |
| E/A | 1.4 ± 0.8 | 1.4 ± 0.8 | 1.5 ± 0.7 | 0.755 |
| EDT | 229 ± 70 | 223 ± 63 | 224 ± 87 | 0.364 |
| Med e' | 5 ± 1.5 | 5 ± 1.6 | 5 ± 1.3 | 0.565 |
| Med a' | 8 ± 2.6 | 8 ± 2.7 | 8 ± 2.2 | 0.973 |
| Med S | 7 ± 1.6 | 7 ± 1.7 | 7.1 ± 1.4 | 0.859 |
| Med E/e' | 17 ± 7.4 | 18 ± 8 | 15 ± 5 | 0.09 |
| Lat e' | 8 ± 2.6 | 7.7 ± 2.6 | 9 ± 2.5 | 0.087 |
| Lat a' | 9.6 ± 3.3 | 10 ± 3.5 | 9 ± 3 | 0.421 |
| Lat S | 8.4 ± 2.3 | 8.6 ± 2.5 | 8 ± 1.7 | 0.190 |
| Lat E/e' | 11 ± 5.4 | 12 ± 6 | 8.4 ± 2 | 0.019 |
| R to AVC | 375 ± 70 | 364 ± 62 | 397 ± 83 | 0.163 |
| SAM | 30 (56%) | 26 (62%) | 4 (28%) | 0.061 |

p-value is between HCM with & without VA groups

***Echocardiography parameters in the HCM cohort***

Interventricular Septum thickness in diastole is 20 ± 6.2 mm, and posterior wall thickness in diastole is 12.3± 3.5 mm. LV Mass Index is ($gm/m^2$) 154± 53 which is higher than for normal individuals as expected in HCM. LV EF by the Simpson method is 61% ±6.2. LAESVi is 33.7 $ml/m^{2,}$ which is on the higher side of the normal value. PW dopplers across mitral valve E is 84 ± 28 mm/sec, and A is 69 ± 31 mm/sec E/A is 1.4 ± 0.8 mm/sec, EDT is 229 ± 70 ms; all are within normal limits, Lateral e' is 8 ± 2.6 mm & medial e' is 5 ± 1.5 mm less than normal values suggestive of LV diastolic dysfunction. Lat E/e' is 11 ± 5.4 Medial E/e' is 17 ± 7.4. Average E/e' is 14, suggestive of LV diastolic dysfunction. SAM is seen in 30 (56%)

patients. Mitral regurgitation is seen in 44 (79%) of patients. The mean LVOT gradient is 33 mm Hg.

***Comparing HCM without and with VA***

The mean LVOT gradient in HCM without VA is 40 mm Hg against HCM with VA of 14 mm Hg, and the LVOT gradient with Valsalva in HCM without VA is 60 mm Hg against HCM with VA of 25 mm statistically significant Hg ($p < 0.05$). HCM patients with VA had a low LVOT gradient compared to HCM patients without VA, so the LVOT gradient does not correlate with the risk of Arrhythmia. The rest of the Echocardiography parameters were similar in both groups**.**

**Strain Echocardiography findings**

***Table 8: Segmental Strain in HCM patients & both the groups***

| | HCM (n=56) | HCM without VA(n=42) | HCM with VA (n = 14) | P |
|---|---|---|---|---|
| Basal Anterior | -13.1 ± 7.6 | -13.6 ± 7.7 | -11.8 ± 7.4 | 0.47 |
| Basal Antero-septal | -9.3 ± 7 | -8.9 ± 7.5 | -10.4 ± 5.2 | 0.51 |
| Basal Antero lateral | -16 ± 7 | -15.7 ± 7.6 | -16.8 ± 6.7 | 0.64 |
| Basal Inferior | -12 ± 7.8 | -11.8 ± 8.4 | -12.7 ± 5.7 | 0.71 |
| Basal Infero septal | -11.6 ± 7 | -11.7 ± 6.9 | -11.3 ± 4.7 | 0.86 |
| Basal Infero lateral | -15 ± 6 | -14.9 ± 5.8 | -15.4 ± 5.9 | 0.81 |
| Mid Anterior | -17 ± 8 | -18.4 ± 6.8 | -14 ± 10.9 | 0.79 |
| Mid Antero-septal | -14 ± 5 | -14.1 ±4.9 | -13.2 ± 3.7 | 0.50 |
| Mid Antero lateral | -17 ± 6 | -17.9 ± 5.8 | -15.4 ± 4.8 | 0.15 |
| Mid Inferior | -15 ± 5 | -15.1 ± 5.7 | -14.4 ± 3.4 | 0.63 |
| Mid Infero septal | -15 ± 5 | -15.7 ± 4.8 | -12.7 ± 3 | **0.038** |

| | | | | |
|---|---|---|---|---|
| Mid Infero lateral | -16.6 ± 9.4 | -16.1 ± 6.3 | - 18.1 ± 15.6 | 0.50 |
| Apical Anterior | - 23 ± 7 | -23 ± 7.6 | -22.8 ± 7.1 | 0.87 |
| Apical Antero-septal | -20.6 ± 6 | -21.1 ± 6.5 | -19.1 ± 4.9 | 0.3 |
| Apical Antero lateral | -21 ± 6 | -22 ± 6.8 | -20 ± 5 | 0.28 |
| Apical Inferior | -22 ± 7 | -22.8 ± 7.5 | -21.1 ± 6.5 | 0.44 |
| Apical Infero septal | -22 ± 6 | -23.5 ± 6.7 | -18.6 ± 4.4 | **0.04** |
| Apical Infero lateral | -17 ± 6 | -18.5 ± 6.9 | -14.3 ± 3.8 | **0.009** |

p-value is between HCM with & without VA groups

Segmental strain values were lower in HCM with VA compared to HCM without VA, but the difference was statistically significant only in mid-inferoseptal, apical inferoseptal, and apical inferolateral segments.

**Global longitudinal Strain (GLS)**

***Table 9: Global longitudinal strain in HCM patients & both the groups***

| | HCM (n=56) | HCM without VA(n=42) | HCM with VA (n = 14) | P |
|---|---|---|---|---|
| Apical 2 Chamber | -17.5 ± 5 | -17.7 ± 5.4 | -16.8 ± 4.5 | 0.58 |
| Apical 3 Chamber | -15.5 ± 4 | -15.8 ± 4.4 | -14.4 ± 3.9 | 0.29 |
| Apical 4 Chamber | -16 ± 8 | -17.4 ± 7.3 | -11.4 ± 11 | 0.027 |
| GLS | -16.8 ± 4.4 | -17.2 ± 4.6 | -15.6 ± 3.5 | 0.249 |

p-value is between HCM with & without VA groups

Strain values in the Apical 2 Chamber, Apical 3 Chamber, Apical 4 chamber, and GLS were lower in HCM with VA compared to HCM without VA. Still, the difference was statistically significant in only the Apical 4 chamber view.

The GLS of the whole HCM cohort is -16.8 ± 4.4. GLS in HCM with VA is -15.6 ± 3.5 compared to -17.2 ± 4.6 in HCM without VA.

**Time to peak strain values**

***Table 10: Time to peak strain in HCM patients & both the groups***

| | HCM (n=56) | HCM without VA(n=42) | HCM with VA (n = 14) | P |
|---|---|---|---|---|
| Basal Anterior | 328 ± 91 | 322 ± 87 | 347 ± 012 | 0.367 |
| Basal Antero-septal | 334 ± 116 | 330 ± 118 | 344 ± 144 | 0.701 |
| Basal Antero lateral | 327 ± 82 | 331 ± 69 | 318 ± 155 | 0.616 |
| Basal Inferior | 328 ± 80 | 319 ± 73 | 355 ± 95 | 0.142 |
| Basal Infero septal | 324 ± 84 | 330 ± 78 | 308 ± 102 | 0.422 |
| Basal Infero lateral | 349 ± 70 | 341 ± 67 | 373 ± 76 | 0.140 |
| Mid Anterior | 347 ± 65 | 336 ± 54 | 381 ± 85 | **0.024** |
| Mid Antero-septal | 358 ± 72 | 349 ± 67 | 386 ± 80 | 0.102 |
| Mid Antero lateral | 342 ± 62 | 340 ± 57 | 352 ± 79 | 0.533 |
| Mid Inferior | 345 ± 63 | 340 ± 58 | 363 ± 76 | 0.244 |
| Mid Infero septal | 343 ± 62 | 335 ±57 | 368 ± 72 | 0.084 |
| Mid Infero lateral | 353 ± 73 | 342 ± 70 | 385 ± 78 | **0.059** |
| Apical Anterior | 338 ± 68 | 331 ± 65 | 361 ± 74 | 0.156 |
| Apical Antero-septal | 345 ± 71 | 337 ± 69 | 369 ± 70 | 0.143 |
| Apical Antero lateral | 332 ± 70 | 321 ± 61 | 363 ± 87 | **0.052** |
| Apical Inferior | 341 ± 67 | 329 ± 60 | 375 ± 78 | **0.025** |

| | | | | |
|---|---|---|---|---|
| Apical Infero septal | 328 ± 67 | 320 ± 63 | 354 ± 75 | 0.107 |
| Apical Infero lateral | 340 ± 62 | 334 ± 61 | 357 ± 66 | 0.232 |

p-value is between HCM with & without VA groups

Time to peak strain was more in HCM with VA compared to HCM without VA, but the difference was statistically significant in mid anterior, mid inferolateral, apical anterolateral, apical inferior segments

Mechanical Dispersion (MD)

***Table 11: Mechanical Dispersion in HCM patients & both the groups***

| | HCM (n=56) | HCM without VA(n=42) | HCM with VA (n = 14) | P |
|---|---|---|---|---|
| Apical 2 Chamber | 56 ± 37 | 57 ± 39 | 54 ± 35 | 0.779 |
| Apical 3 Chamber | 57 ± 56 | 53 ± 40 | 69 ± 88 | 0.370 |
| Apical 4 Chamber | 58 ± 38 | 56 ± 39 | 65 ± 34 | 0.461 |
| MD | 43.7 ± 23 | 41 ± 26 | 51 ± 39 | 0.249 |

p-value is between HCM with & without VA groups

Mechanical dispersion (Standard deviation of time to peak strain from all 18 segments) of the HCM cohort is 43.7 ± 23. MD in HCM with VA group is 51± 39 ms and 41 ± 26 in HCM without VA group. MD was more in HCM with VA than HCM without VA; however, it was statistically insignificant.

**MRI findings**

MRI was done in 44 (78%) of 56 HCM patients, 33 (78%) of 42 HCM without VA, and 11 (78%) of 14 HCM with VA patients.

***Table 12: MRI wall thickness in HCM patients & both the groups***

| | HCM (n=44) | HCM without VA(n=33) | HCM with VA (n = 11) | P |
|---|---|---|---|---|
| Basal Anterior | 15.5 ± 7 | 15.5 ± 7 | 15.7 ± 7 | 0.945 |
| Basal Antero-septal | 19.6 ± 5.3 | 20 ± 5.6 | 18.5 ± 4.3 | 0.451 |
| Basal Antero lateral | 10.1 ± 4.4 | 10.1 ± 4.2 | 10.2 ± 5.6 | 0.991 |
| Basal Inferior | 12.5 ± 5.7 | 12.8 ± 6 | 11.8 ± 4 | 0.601 |
| Basal Infero septal | 15.8 ± 5.1 | 15.9 ± 4.3 | 15.5 ± 7.1 | 0.829 |
| Basal Infero lateral | 9 ± 3.6 | 8.9 ± 3.6 | 9.3 ± 3.7 | 0.716 |
| Mid Anterior | 14 ± 4.4 | 13.3 ± 4.3 | 16 ± 4.2 | 0.081 |
| Mid Antero-septal | 17.8 ± 6.4 | 17.5 ± 7.2 | 18.5 ± 3.1 | 0.674 |
| Mid Antero lateral | 10.5 ± 4.4 | 9.8 ± 3.9 | 12.8 ± 5.2 | **0.05** |
| Mid Inferior | 14.4 ± 5.5 | 13.8 ± 5.5 | 16.1 ± 5.1 | 0.233 |
| Mid Infero septal | 17.7 ± 6.3 | 16.8 ± 6.3 | 20.3 ± 5.5 | 0.108 |
| Mid Infero lateral | 10.2 ± 3.9 | 9.8 ± 3.8 | 11.1 ± 4.2 | 0.374 |
| Apical Anterior | 12.4 ± 5.2 | 11.8 ± 5.3 | 14.1 ± 4.4 | 0.212 |
| Apical septal | 13 ± 5.9 | 12 ± 5.6 | 16.1 ± 5.6 | **0.041** |
| Apical lateral | 11.5 ± 4.8 | 10.9 ± 4.8 | 13.5 ± 4.4 | 0.118 |
| Apical Inferior | 12.3 ± 5.4 | 11.5 ± 5.3 | 14.9 ± 5.1 | 0.069 |
| Apical | 10.7 ± 5.5 | 10.4 ± 5.7 | 11.6 ± 5 | 0.542 |
| MWT | 22.6 ±5 | 22.6 ± 5.2 | 22.8 ± 4.7 | 0.892 |

p-value is between HCM with & without VA groups

MRI segmental wall thickness was similar in both the groups, except for mid-anterolateral & apical septal segments in which HCM with VA has higher thickness than HCM without VA, which was statistically significant.

**LGE & LGE volume %**

***Table 13: MRI LGE & LV mass in HCM patients & both the groups***

| | HCM (n=44) | HCM without VA(n=33) | NSVT (n = 11) | P |
|---|---|---|---|---|
| LGE | 35 (79%) | 24 (72%) | 11 (100%) | **0.05** |
| Myocardial volume (ml) | 122 ± 53 | 121.8 ± 57 | 122.7 ± 11 | 0.964 |
| LGE volume (ml) | 18.7 ± 16 | 18 ± 18 | 20 ± 11 | 0.729 |
| LGE volume % | 12.9 ± 8.4 | 13.5 ± 9 | 11.5 ± 7 | 0.531 |
| Myocardial mass (gm) | 128 ± 56 | 127 ± 60 | 128 ± 50 | 0.969 |
| LGE mass (gm) | 23.8 ± 30 | 25 ± 25 | 21 ± 11 | 0.731 |
| LV diastole mass (gm) | 142 ± 57 | 140 ± 59 | 150 ± 53 | 0.699 |
| LV diastole mass indexed (gm/m2) | 85 ± 32 | 84 ± 34 | 88 ± 27 | 0.806 |

p-value is between HCM with & without VA groups

LGE on MRI was present in 35 (79%) patients in 44 patients who underwent MRI. 24 (72%) patients in HCM without VA had LGE on MRI, whereas 11 (100%) patients had LGE on MRI in HCM with VA group which is statistically significant.

Myocardial volume, LGE volume, LGE volume %, Myocardial mass & LGE mass were similar in both the groups.

***Table 14: Segmental LGE in HCM patients & both the groups***

| | HCM LGE (n=35) | HCM without VA (n=24) | HCM with VA (n = 11) | p-0.05 |
|---|---|---|---|---|
| Basal Anterior | 9 (25%) | 6 (25%) | 3 (27%) | 0.886 |
| Basal Antero-septal | 16 (45%) | 11 (45%) | 5 (45%) | 0.824 |
| Basal Antero lateral | 4 (11%) | 2 (8%) | 2 (18%) | 0.395 |
| Basal Inferior | 4 (11%) | 4 (16%) | 0 | 0.150 |
| Basal Infero septal | 10 (28%) | 6 (25%) | 4 (36%) | 0.490 |
| Basal Infero lateral | 2 (5%) | 2 (8%) | 0 | 0.313 |
| Mid Anterior | 14 (40%) | 9 (37%) | 5 (45%) | 0.656 |
| Mid Antero-septal | 21 (60%) | 14 (58%) | 7 (63%) | 0.766 |
| Mid Antero lateral | 11 (31%) | 7 (29%) | 4 (36%) | 0.670 |
| Mid Inferior | 6 (17%) | 5 (20%) | 1 (9%) | 0.392 |
| Mid Infero septal | 20 (57%) | 14 (58%) | 6 (54%) | 0.833 |
| Mid Infero lateral | 3 (8%) | 2 (8%) | 1 (9%) | 0.941 |
| Apical Anterior | 15 (42%) | 8 (33%) | 7 (63%) | 0.093 |
| Apical septal | 11 (31%) | 4 (16%) | 7 (63%) | **0.05** |
| Apical lateral | 12 (34%) | 5 (20%) | 7 (63%) | **0.013** |
| Apical Inferior | 8 (22%) | 4 (16%) | 4 (36%) | 0.198 |
| Apical | 13 (52%) | 5 (20%) | 8 (72%) | **0.003** |

p-value is between HCM with & without VA groups

LGE was present in all patients of HCM with VA, whereas 24 (72%) patients had LGE in HCM without VA group, which was statistically significant. Segmental LGE was similar among both groups except for Apical septal, Apical lateral, and Apical segments, where LGE was present more in HCM with VA compared to HCM without VA group and was statistically significant.

**Global longitudinal Strain & Mechanical dispersion in patients with LGE on MRI**

***Table 15: Segmental Strain in Patients with LGE (n=35) on MRI***

| Segmental Strain | HCM without VA (n=24) | HCM with VA (n=11) | p |
|---|---|---|---|
| Basal Anterior | -12.5 ± 7.9 | -10.6 ± 7.2 | 0.254 |
| Basal Antero-septal | -9.5 ± 6.3 | -10.6 ± 5.4 | 0.309 |
| Basal Antero lateral | -13.8 ± 8.4 | -15.4 ± 6.5 | 0.286 |
| Basal Inferior | -10.4 ± 8.6 | -12.1 ± 6 | 0.276 |
| Basal Infero septal | -10.6 ± 7.9 | -10.3 ± 4 | 0.459 |
| Basal Infero lateral | -14 ± 6 | -14.7 ± 6 | 0.391 |
| Mid Anterior | -17.5 ± 7.2 | -11.3 ± 10 | **0.025** |
| Mid Antero-septal | -13.4 ± 4.7 | -12.5 ± 3.8 | 0.295 |
| Mid Antero lateral | -16.5 ± 6 | -14.6 ± 5.1 | 0.179 |
| Mid Inferior | -14.6 ± 5.9 | -14.4 ± 3.4 | 0.441 |
| Mid Infero septal | -15.7 ± 5.3 | -11.1 ± 2.6 | **0.019** |
| Mid Infero lateral | -16.2 ± 6.2 | -14.1 ± 5 | 0.162 |
| Apical Anterior | -22.4 ± 7.4 | -21 ± 6.3 | 0.293 |
| Apical Antero-septal | -19.8 ± 6.3 | -18.6 ± 4.9 | 0.284 |
| Apical Antero lateral | -22 ± 7 | -18.7 ± 5 | 0.079 |
| Apical Inferior | -22.4 ± 8 | -18.5 ± 3.8 | 0.071 |

| | | | |
|---|---|---|---|
| Apical Infero septal | -23.2 ± 7.3 | -17.4 ± 3.5 | **0.009** |
| Apical Infero lateral | -17.9 ± 7 | -15 ± 4.1 | 0.105 |

Segmental strain values were lower in HCM with VA compared with HCM without VA group; however, they were statistically insignificant, except for Mid Anterior, Mid Infero septal, Apical Infero septal, where the difference in segmental strain was statistically significant.

***Table 16: Global longitudinal Strain in Patients with LGE (n=35) on MRI***

| Segmental Strain | HCM without VA n=24 | HCM withVA n=11 | P |
|---|---|---|---|
| Apical 2 Chamber | -16.9 ± 5.9 | -15.7 ± 4.4 | 0.270 |
| Apical 3 Chamber | -15.5 ± 4.7 | -14.3 ± 4.1 | 0.259 |
| Apical 4 Chamber | -16.9 ± 5.7 | -9 ± 11 | **0.006** |
| GLS | -16.4 ± 5.1 | -14.7 ± 3.3 | 0.167 |

AP2CV, AP3CV& GLS are lower in HCM with VA compared to HCM without VA but statistically insignificant. AP4CV strain was lower in HCM with VA compared to HCM without VA and was statistically significant.

***Table 17: ESC 5-year SCD risk score***

| | HCM (n=56) | HCM without VA(n=42) | HCM with VA (n = 14) | P |
|---|---|---|---|---|
| | 3.9 ± 3.1 | 3.1 ± 2.5 | 6.6 ± 3.5 | < **0.01** |

p-value is between HCM with & without VA groups

ESC 5-year SCD risk score was higher in HCM with VA than in HCM without VA, which is statistically significant.

***Table 18: AHA/ACC SCD Risk Markers***

| | HCM (n=56) | HCM without VA(n=42) | HCM with VA (n = 14) | P |
|---|---|---|---|---|
| Family History of HCM-related SCD | 6 (11%) | 4 (9%) | 2 (14%) | 0.618 |
| Unexplained Syncope | 19 (33%) | 11 (26%) | 8 (57%) | **0.034** |
| NSVT | 14 (25%) | 0 | 11 (78%) | **< 0.01** |
| Left Ventricular Hypertrophy (LVH) ≥ 30 mm | 7 (12%) | 6 (14%) | 1 (7%) | 0.484 |
| LGE > 15% LV volume | 13 (23%) | 9 (21%) | 4 (28%) | 0.877 |
| LV Apical Aneurysm | 0 | 0 | 0 | - |
| Ejection fraction < 50% | 0 | 0 | 0 | - |

p-value is between HCM with & without VA groups

Unexplained Syncope is more common in HCM with VA than in HCM without VA group, which is statistically significant. None of the patients had LV apical aneurysms on Echocardiography or MRI. All patients with less than 55% ejection fraction were excluded from the study. Family history of HCM-related SCD, Left ventricular hypertrophy > 30 mm, and LGE > 15% LV volume is similar in both groups.

**ROC Curves for diagnosing Ventricular Arrhythmia in HCM**

**Mechanical Dispersion**

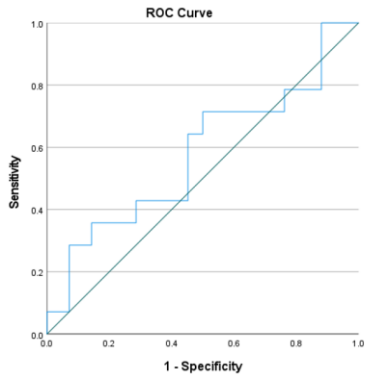


***Figure 1: ROC curve – Mechanical dispersion to diagnose HCM with VA***

AUC for Mechanical dispersion to diagnose HCM with VA is .578.

**GLS**

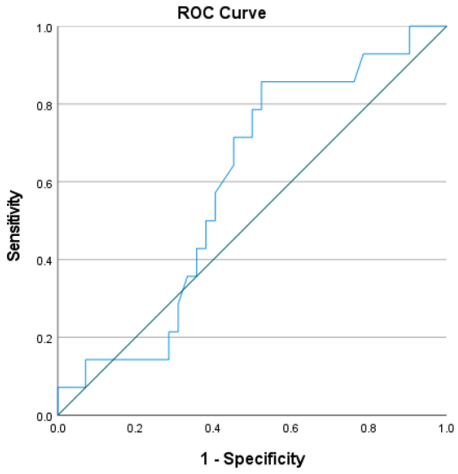


***Figure 2: ROC curve - GLS to diagnose HCM with VA***

AUC for GLS to diagnose HCM with VA is .592

**LGE Volume % on MRI**

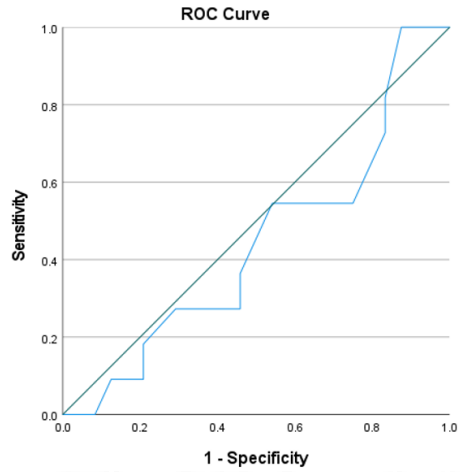


***Figure 3: ROC curve – LGE Volume % to diagnose HCM with VA***

AUC for LGE Volume %  to diagnose HCM with VA is 0.441

**Syncope**

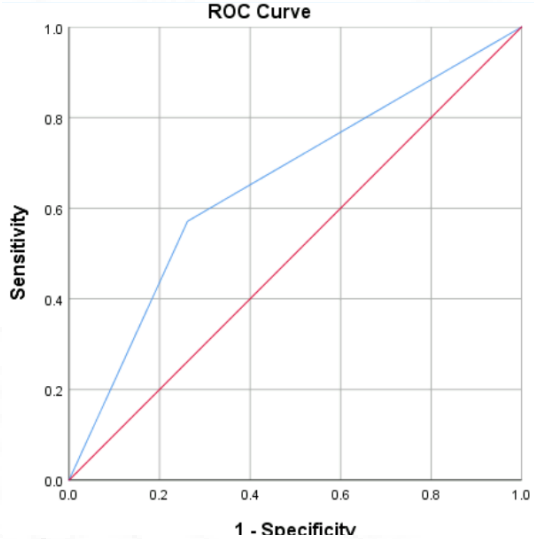


***Figure 4: ROC curve- Syncope to diagnose HCM with VA***

AUC for Syncope to diagnose HCM with VA is 0.655

**ESC 5-year SCD risk score**

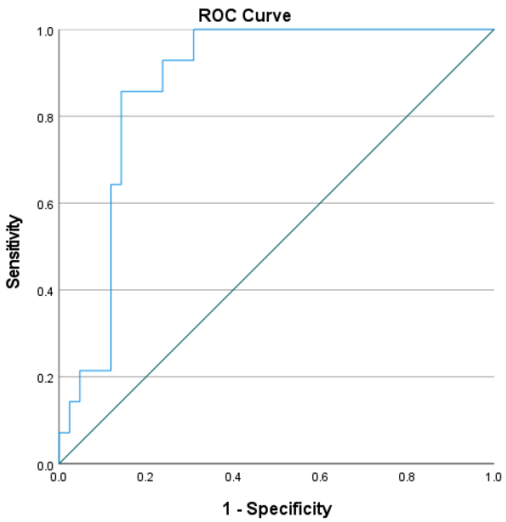


***Figure 5: ROC curve* - ESC 5-year SCD risk score to diagnose HCM with VA**

AUC for ESC 5-year SCD risk score to diagnose HCM with VA is .874

With ROC curves, among the various variables of GLS, MD, % LGE on MRI, Syncope & ESC 5-year SCD risk score, only ESC 5-year SCD risk score has significant AUC (0.874) to diagnose HCM patients with VA.

# DISCUSSION

In our cohort of HCM patients, 68% are male patients and 93% in HCM with VA group. Though male predominance of the HCM cohort is found in other studies, strikingly 93% male predominance in HCM with VA is not found in other studies (Haland et al., 2016) (Candan et al., 2017). The majority of HCM without VA group 64% are NYHA Class II. In contrast, 71% of patients with HCM with VA are NYHA Class I, signifying there is no correlation between symptoms & having VA & SCD, and asymptomatic patients may present with SCD. Dyspnea in 40% of patients is the most common symptom in the whole cohort, followed by Syncope in 34%. Syncope is the most common symptom in HCM with VA group, the presence of Syncope is the most critical factor in determining the risk of Arrhythmia in our study ($p<0.05$).

The family history of HCM is present in only 20% of our cohort, and the family history of SCD in 11% of patients. LVOT gradient is low 14 mm Hg in HCM with VA compared to 40 mmHg in HCM without VA ($P < 0.05$); hence, most patients with HCM with VA have no LVOTO.

Medial e' and lateral e' in our cohort are 5 & 8 cm/sec, implying diastolic dysfunction in all the patients in both groups. Segmental strain values, AP2CV strain, AP3CV strain, and GLS are lower in HCM with VA compared with HCM without VA; however, the difference was not statistically significant except for AP4CV strain, & segmental strain mid-inferoseptal, apical inferoseptal, apical inferolateral where the difference was statistically significant. Overall GLS of our HCM cohort is – 16.8 ± 4.4 compared to -15.7 ± 3.6 (Haland et al., 2016), -12.1 ± 3.4 (Candan et

al., 2017), which is slightly higher in our study than studies mentioned above. GLS in HCM patients is lower than in control subjects -21.9 ± 1.9 (Haland et al., 2016), -20.7 ± 2.4 (Jalanko et al., 2016), -20 ± 2.5 (Popa-Fotea et al., 2020) in various other studies. GLS in HCM with VA group in our study is 15.6 ± 3.5 compared to -14.1 ± 3.6 (Haland et al., 2016), -14.7 ± 4.1 (Jalanko et al., 2016), −11.85 ± 5.7 (Popa-Fotea et al., 2020), -10.6 ± 2.8(Candan et al., 2017) while GLS in HCM without VA in our group is -17.2 ± 4.6 compared with -16.3 ± 3.4 (Haland et al., 2016), -17.4 ± 3.8 (Jalanko et al., 2016), −15.63 ± 4.7 (Popa-Fotea et al., 2020), -12.7 ± 3.5 (Candan et al., 2017) as seen GLS is lower in HCM patients with VA compared to without VA.

Mechanical dispersion in our HCM cohort is 43.7 ± 30 ms, which is less than compared with other studies, 64 ± 22 ms (Haland et al., 2016) and 66.4 ± 19.4 ms (Candan et al., 2017). MD in normal subjects in other studies is 36 ± 13 ms (Haland et al., 2016), 41 ± 16 ms (Jalanko et al., 2016), and 39 ± 12 ms (Popa-Fotea et al., 2020).

MD in HCM with VA in our group is 51.8 ± 39 ms compared to 79 ± 27 ms (Haland et al., 2016), 93 ± 41ms (Jalanko et al., 2016), 77.1 ± 28 ms (Candan et al., 2017), 81 ± 18 ms (Popa-Fotea et al., 2020), MD in HCM without VA group in our study is 41.04 ± 26 ms compared with 59 ± 16 ms (Haland et al., 2016), 50 ± 18 ms (Jalanko et al., 2016), 62.4 ± 17.1 ms (Candan et al., 2017), 42 ± 8 ms (Popa-Fotea et al., 2020).

As a trend, GLS is lower in HCM patients than in normal subjects. HCM with VA still has lower starin values than HCM without VA. MD is pronounced in HCM

patients compared to normal subjects. HCM with VA has more pronounced MD compared to HCM without VA.

***Table 19: GLS and MD in our cohort & various other studies categorized into HCM with and without VA***

| | **GLS** | | | | **MD** | | | |
|---|---|---|---|---|---|---|---|---|
| | HCM | HCM without VA | HCM with VA | Healthy control | HCM | HCM without VA | HCM with VA | Healthy control |
| Our data SCTIMST Philips EPIQ 7c (Q Lab) | -16.8 ± 4.4 (56) | -17.2 ± 4.6 (42) | -15.6 ± 3.5 (14) | | 43.7 ± 30 | 41.04 ± 26 | 51.8 ± 39 | |
| Haland etal 2016 GE (EchoPAC) | -15.7 ± 3.6 (150) | -16.3 ± 3.4 (113) | -14.1 ± 3.6 (37) | -21.9 ± 1.9 (50) | 64 ± 22 | 59 ± 16 | 79 ± 27 | 36 ± 13 |
| Jalanko etal 2016 GE (EchoPAC) | - (31) | -17.4 ± 3.8 (20) | -14.7 ± 4.1 (11) | -20.7 ± 2.4 (20) | - | 50 ± 18 | 93 ± 41 | 41 ± 16 |
| Candan etal 2017 GE (EchoPAC) | -12.1 ± 3.4 (63) | -12.7 ± 3.5 | -10.6 ± 2.8 | - | 66.4 ± 19.4 | 62.4 ± 17.1 | 77.1 ± 28 | - |
| Fotea etal 2020 GE (EchoPAC) | | −15.63 ± 4.7 (31) | −11.85 ± 5.7 (16) | -20 ± 2.5 (36) | | 42 ± 8 | 81 ± 18 | 39 ± 12 |

A lot of difference in GLS & MD in various HCM patients in various above studies, including ours, is due to different Echo machines & software used to analyze strain. We analyzed strain using Philips EPIQ 7c with Q Lab. The effect of various software vendors on GLS in HCM was studied by Sperry et al. (Sperry et al.,

n.d.) where they did strain on the same HCM patients using three different software and found GLS of −20.8±2.7 with Philips EPIQ 7c using Q Lab, −15.7±2.7 with Siemens using Velocity Vector Imaging, and −16.1±3.2 with GE Medical using EchoPAC.

Higher values of GLS in our study might be related to using Philips EPIQ 7c as correlated with the above study.

The effect of various software vendors on MD in HCM was studied by Saijo et al. (Saijo et al., 2020), where they did MD on the same HCM patients using three different software and found MD of 52 ± 21 ms with Philips EPIQ 7c using Q Lab, 62 ± 21ms with Siemens using Velocity Vector Imaging, 62 ± 24 with GE Medical using EchoPAC Lower MD values in our study might be related to us using Philips EPIQ 7c as correlated with above study.

***Table 20: GLS & MD in our study and by other Vendors with different software***

| | **GLS** | **MD** |
|---|---|---|
| Our data - SCTIMST<br>Philips EPIQ 7c<br>(Q Lab) | -16.8 ± 4.4 | 43.7 ± 30 |
| **Saijo etal 2019**<br>Philips EPIQ 7c<br>(Q Lab) | | 52 ± 21 |
| Siemens<br>VVI | | 62 ± 21 |
| GE<br>(EchoPAC) | | 62 ± 24 |
| Sperry etal 2019<br>Philips EPIQ 7c<br>(Q Lab) | −20.8±2.7 | |
| Siemens | −15.7±2.7 | |

| | | |
|---|---|---|
| VVI | | |
| GE (EchoPAC | −16.1±3.2 | |

Though there was a correlation between MD & risk of VA in other studies, we did not find a statistically significant correlation between MD & risk of VA in our study.

**LIMITATIONS OF OUR STUDY**

1) Small sample size
2) MRI was not done on all the patients

## SUMMARY AND CONCLUSIONS

- We studied 56 HCM patients and categorized them into two groups, 42 into HCM without VA and 14 into HCM with VA
- 68% of HCM patients are male, 60% in HCM without VA & 93% in HCM with VA
- 82% had Asymmetrical Septal hypertrophy & 12.5 % had Apical hypertrophy
- Baseline characteristics are similar across both groups except for sex
- ECG parameters are similar across both groups except for LVH more present in HCM without VA ($p<0.05$)
- On Holter, 11 patients had NSVT, average heart rate, APC %, and VPC % are similar across both the groups
- Syncope is the most common symptom in HCM in VA group (57%), whereas dyspnea is the most common symptom in HCM without VA group (45%).
- F/h/o HCM & F/h/o SCD is similar across both the groups
- 82% of patients are on beta-blockers which was similar across both the groups
- 93% (13 patients) in HCM with VA underwent ICD implantation compared to 9.5% (4 patients) in HCM without VA
- On Echocardiography parameters, HCM with VA has a low LVOT grad of 14 mm Hg compared to 40 mm hg in HCM without VA group ($p < 0.05$), the rest of the echo parameters are similar across both the groups
- GLS in the HCM cohort is -16.8 ± 4.4, GLS of HCM without VA is 17.2 ± 4.6, and GLS of HCM with VA is -15.6 ± 3.5. Overall GLS of the whole cohort is less than the normal GLS ( -20 to - 22) seen in the control population in other studies, implying that HCM patients have a lower GLS value than normal

patients. GLS is further low in HCM with VA compared to HCM without VA, as seen in other studies. However, our study's difference in GLS between HCM with VA & HCM without VA is statistically insignificant (p – 0.249). (Haland et al., 2016), (Jalanko et al., 2016) (Candan et al., 2017) (Popa-Fotea et al., 2020)

- MD in the HCM cohort is 43.7 ± 23 ms, MD of HCM without VA is 41 ± 26, and MD of HCM with VA is 51 ± 39. MD was more in HCM with VA compared to HCM without VA
- Mechanical dispersion difference is statistically not significant between HCM with VA & HCM without VA (p-0.249)
- Presence of LGE on MRI ($p < 0.05$), Syncope ($p < 0.05$) , ESC 5 year SCD risk scores ($p < 0.05$) were statistically different in HCM with VA & HCM without VA
- Among LGE on MRI, Syncope, and ESC 5-year SCD risk score, only ESC 5-year SCD risk score was able to diagnose HCM with VA with AUC 0.874 in the ROC curve.

**CONCLUSION**

There is no correlation between Mechanical dispersion & Ventricular Arrhythmia in HCM patients in our study.

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